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VITAL ACTIONS ARE ARRESTED IN ASPHYXIA.

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ORDER OF SUCCESSION IN WHICH THE VITAL ACTIONS ARE ARRESTED IN ASPHYXIA.*

A knowledge of the order of succession in which the vital actions of the body are brought to a stand in asphyxia, is not only useful in elucidating the exact nature of the function of respiration, and in pointing out rules for our guidance in the direction of certain remedial agents, but it may also be brought to bear in an important manner upon the investigations into the general laws of physiology. The inquiry is one, however, of unusual difficulty, from the intimate manner in which the respiration is associated in the higher animals with the other vital functions, and the rapidity and energy of the actions and reactions of these upon each other. In conducting such experiments, it is not only necessary to watch closely every phenomenon which presents itself, however fleeting it may be, but all the varied concomitant circumstances must also be carefully analysed, and, if possible, insulated, with the view of ascertaining how far they might affect the results. Our progress in such investigations must, therefore, always necessarily be slow, frequently vacillating and uncertain. These difficulties were found so perplexing, that I had several times nearly given up the present inquiry in despair; and it was not without much labour, and repeated failures, that I arrived at what I considered satisfactory results.

The two points in the physiology of asphyxia which have of late years principally attracted attention are, the nature of the impediment to the circulation of the blood through the lungs; and the cause of the arrestment of the sensorial functions. A correct knowledge of the manner in which the vital actions are arrested in asphyxia, is supposed to be included in the true explanations of these two facts. The first of these, viz. the impediment to the free passage of the blood through the vessels of the lungs, and its consequent stagnation in the right side of the heart, and the large vessels leading to that organ, have been attributed to three causes,—the cessation of the mechanical movements of the chest; the effects of the venous blood upon the contractility of the heart; and the difficulty of transmitting the venous blood through the capillaries of the lungs, when the chemical changes which go on there between the blood and the atmospheric air have ceased. The opinion, that the blood in death from asphyxia chiefly accumulates

* An epitome of this paper was read at last meeting of the British Scientific Association.

in the right side of the heart and the large vessels leading to it, in consequence of the stoppage of the mechanical movements of the chest, was advocated by Haller.* He maintained, that when the lungs were distended with air, as in inspiration, that the blood flowed readily and abundantly through the pulmonary vessels; but, on the other hand, when these organs had collapsed, as in expiration, the pulmonary blood-vessels were so compressed, and their angles rendered so acute, that they became in a great measure impermeable to the blood sent from the right side of the heart.† Goodwyn‡ argued, in opposition to the mathematical calculations and reasonings adduced by Haller, that, when the lungs are diminished in their bulk, and the acuteness of the angles of the blood-vessels changed only to the extent which occurs during expiration, the flow of blood through them would not be materially obstructed. He also drew additional arguments in favour of this opinion, from the continuance of the circulation through the lungs, when an amount of fluid was present in the chest sufficient to compress the lung to the extent which occurs in expiration, whether this fluid had been effused in the human species from disease, or induced by artificial means in the lower animals.§ Goodwyn maintained that the cessation of the circulation in asphyxia was chiefly dependent upon the venous blood failing to excite the contractions of the left side of the heart. "When respiration," he says, "is obstructed, the florid colour of the blood is gradually diminished, and the contractions of the left auricle and ventricle soon cease. The cessation of contraction arises from a defect of a stimulating quantity in the blood itself."‖ The views of Goodwyn were attacked a few years after their promulgation by Coleman¶ and Kite.** Both these authors adduced the results of various experiments, to prove that the left side of the heart can contract vigorously upon venous blood;†† and they also both maintained that they had proved experimentally, that, when the lungs are kept mechanically distended during the process of asphyxia, that the quantity of blood found in the right side after death is not found to preponderate much, if at all, over that contained in the left side.‡‡ Bichat also furnished abundant evidence to prove that the left side of the heart can contract vigorously upon venous blood. In nu-

* Elementa Physiologiæ, Tom. iii. Lib. viii. Sect. iv.

† Opus cit. Tome iii. p. 246. 1776.

‡ The Connection of Life with Respiration. London, 1788.

§ Opus cit. p. 40–47.

‖ Opus cit. p. 85.

¶ A Dissertation on Suspended Respiration, 1791.

** Essays and Observations, &c. on the Submersion of Animals, &c. 1795.

†† Coleman, Opus cit. p. 118; and Kite, Opus cit. p. 26, 42, and 44.

‡‡ Coleman, p. 107 to 116; Kite, "From these experiments, it is evident that a small quantity of blood can pass through the lungs when they are in a state of perfect expiration."—P. 58.

merous experiments he found, that when an animal is asphyxiated, black blood at first traverses the lungs to reach the left side of the heart, and may for a short time be projected from a cut artery, with very considerable force; and he further satisfied himself, that the contractions of the heart could be renewed even after they had become quiescent in different kinds of violent deaths, by injecting venous blood along one of the pulmonary veins towards the left side of the heart.* Bichat especially dwelt upon the importance of discriminating between the effects of asphyxia upon the functions of animal and those of organic life; of ascertaining the priority of the suspension of those two great sets of functions, and the influence which they exerted upon each other. He maintained that the heart's action does not cease because the dark blood transmitted to the left side of the heart cannot excite it to contract, but because the dark blood, by being circulated through the coronary arteries in the muscular tissue of the heart, arrests its contractility. This effect of the dark blood upon the contractility of the heart was, however, regarded by Bichat as only an isolated phenomenon in asphyxia; for he believed that the vitality of all the tissues of the body was equally affected by the circulation of this dark blood, and that the functions of the brain or the animal functions were always arrested before those of organic life.† He maintained that the accumulation of the blood in the right side of the heart did not depend upon any mechanical obstruction in the blood-vessels of the lungs, but from various other causes, among which he enumerates the obstacles opposed to the force of the already enfeebled right side of the heart, by the effects of the circulation of dark blood in the bronchial arteries, and the cessation of the excitation of the lungs by the atmospheric air,‡ aided by the circumstance, that the systemic ventricle can more easily overcome the resistance presented by the capillaries of the body generally, than the veins and pulmonic ventricle can overcome that arising from the capillaries of the lungs. Bichat appears to have entertained doubts whether the circulation of the venous blood through the capillaries of the systemic circulation arrested the vitality of the tissues simply by default of excitation, or by exerting some deleterious influence upon it; for, while discussing its effects upon the brain, he thus expresses himself—"Je ne puis dire si c'est négativement ou positivement que s'exerce son influence; tout ce que je sais, c'est que les fonctions du cerveau sont suspendues par elle." Although Bichat failed in giving the correct explanation of the manner in which the vital actions are arrested in

* Sur La Vie et La Mort, article sixieme, § ii.

† Car, d'après ce que nous dirons, l'affaiblissement qu' éprouve alors le cœur n'est qu'un symptôme particulier de cette maladie dans lequelle tous les autres organes sont le siége d'une semblable débilité.

‡ "Le défaut de son excitation par l'air vital."

asphyxia, yet there can be no doubt that to him we are indebted for having pointed out the true path by which this knowledge was to be attained. Another important advance was made in the elucidation of asphyxia by the experiments of Dr David Williams of Liverpool and Dr J. P. Kay. Dr Williams,* in experimenting on this subject, found " that when the chest is laid open immediately after the trachea has been tied during the acme of inspiration, the pulmonary veins soon become empty, while the pulmonary artery continues full." From these experiments he inferred, that, in asphyxia, the blood is obstructed in its passage through the lungs, while its circulation through the other tissues of the body continues; and that the obstruction in the lungs " arises from a deprivation of pure atmospheric air." Dr Kay, from his numerous experiments,† has also arrived at the conclusion, that " the circulation is arrested after respiration ceases; because, from the exclusion of oxygen, and the consequent non-arterialization of the blood, the minute pulmonary vessels, which usually convey arterial blood, are then incapable of conveying venous blood, which therefore stagnates in the lungs."‡ Dr Kay believes that this stagnation of blood in the right side of the heart and pulmonary artery, occurs in consequence of venous blood being incapable of exciting the arterial capillary blood-vessels of the lungs. The experiments of Dr W. F. Edwards§ upon frogs, and those of Dr Kay upon warm-blooded animals, have very distinctly proved, that the circulation of venous blood in the muscular tissue not only does not exert any deleterious influence upon its contractility, but that this property continues to manifest itself considerably longer when venous blood is allowed to circulate through the vessels of that tissue, than when the circulation of the blood has been entirely arrested.

Though the experiments of Drs Williams and Kay have demonstrated, that in asphyxia the circulation is first brought to a stand by some impediment to its free passage through the lungs, yet we believe that few will feel satisfied, after a careful analysis of them, that they enable us to determine whether this impediment results from the cessation of the respiratory movements of the chest, or from the arrestment of the usual chemical changes between the blood and the atmospheric air,—a question of considerable importance in general physiology. When we remember the great influence exerted by the respiratory muscular movements upon the force with which the blood is transmitted along its vessels, —a fact first well illustrated by Hales,‖ afterwards by Bichat,¶

* On the Cause and the Effects of an Obstruction of the Blood in the Lungs, Edinburgh Medical and Surgical Journal, Vol. xix. p. 524.

† The Physiology, Pathology, and Treatment of Asphyxia, 1834.

‡ Opus cit. p. 181.

§ De L'Influence des Agens Physiques sur la Vie, p. 9, 1824.

‖ Statistical Essays, Vol. ii. p. 1 to 33, 1740.

¶ Sur la Vie et la Mort, article huitième, § ii.

and latterly in a more definite manner by Magendie* and by Poiseuille,† a degree of uncertainty must always exist in interpreting phenomena observed in experiments upon asphyxia, in which means have not been taken to obtain the extent and value of this influence.

Such a precaution is the more necessary, since it has been ascertained that dark blood passes at first in the usual quantity through the lungs, and is sent with great force and in a full stream from a cut artery; that it is not until the respiratory movements have been considerably impaired that it begins to stagnate in the lungs; and that after death, considerable quantities of dark blood are frequently obtained from the left side of the heart. Coleman found that the relative quantity of blood in the two sides of the heart after drowning varied considerably; "sometimes being as 7 to 4, at other times as 5 to 2, or as 12 to 7. So that, at a medium, the proportions of the right one to the left are about $3\frac{2}{8}$ to $1\frac{6}{8}$. After hanging, the medium was found to be as $2\frac{7}{8}$ to $1\frac{4}{8}$.‡

Professor Alison, with the view of supplying this defect in the theory of asphyxia, performed several times the following experiment.§ A rabbit was confined in nitrogen gas until its respiratory movements had become laboured, and insensibility was approaching The animal was then withdrawn as rapidly as possible from the glass jar in which it had been confined, and the brain was suddenly crushed by a blow with a hammer, and the chest was immediately laid open. The quantity of blood found in the right side of the heart preponderated considerably over that in the left, and, as the respiratory movements had not been interrupted until the animal had been deprived of life, and the circulation in a great measure suspended, these experiments are obviously greatly in favour of the opinion, that the accumulation of the blood in and around the right side of the heart is dependent upon the cessation of the chemical changes between the blood and atmospheric air in the lungs, and not upon the arrestment of the mechanical movements of the chest.

It appeared to me that very conclusive evidence might be obtained on this question by a series of experiments performed in the following manner. A tube with a stop-cock was fixed into an opening in the trachea, and one of Poiseuille's hemadynamometers was introduced into the femoral artery, for the purpose of obtaining definite information upon the force with which the blood was transmitted along the arterial system. The stop-cock of the tube

* Journal de Physiologie, Tom. i. Leçons sur les Phénomenes Physiques de la Vie.

† Journal de Physiologie, Tom. viii. p. 272.

‡ Opus cit. p. 18.

§ Edinburgh Medical and Surgical Journal, Vol. xlv. p. 103.

in the trachea was then shut, and when the respiratory process had been suspended sufficiently long to cause a decided fall in the column of mercury supported by the blood sent along the femoral artery, a large bladder full of pure nitrogen with a brass nozzle provided with a stop-cock, was fixed in the tube in the trachea, which it fitted accurately, and both stop-cocks opened. After the effects of the respiration of the nitrogen gas had been ascertained, a bladder of the same size as the other, similarly provided with a nozzle, and full of atmospheric air, was then substituted for the bladder containing the nitrogen, and the results compared. The difference between the effects of the respiration of the nitrogen gas and the atmospheric air was most marked, and of such a nature as could not be mistaken ; for while the mercury continued to fall in the instrument during the respiration of the nitrogen gas, it rose very rapidly immediately after the atmospheric air had entered the lungs and acted upon the blood. In this experiment the same mechanical movements of the chest which failed to renew the free circulation of the blood through the lungs when nitrogen gas was inspired, rapidly effected that object when atmospheric air was permitted to enter the lungs, even when tried on the same animal, and subsequent to the failure of the nitrogen, and, consequently, at a more advanced stage of the process of asphyxia. This experiment was repeated several times, and when the requisite care was taken to procure and employ pure nitrogen, invariably with the same results.*

Before directing the attention of the reader to a table containing the results of one of these experiments, it will be necessary to take notice of a very unexpected phenomenon which presented itself, and for a considerable time completely embarrassed and perplexed me. Before commencing these experiments I conceived from *a priori* reasoning, that when the blood had become dark in the arteries, and the animal functions had been suspended, that the mercury would begin to fall gradually and steadily in the hemadynamometer, and that there would in a short time be a marked depression in the level of the mercury. The mercury, however, actually stood higher in the instrument, and the large arteries became more distended and tense for about two minutes after the animal had become insensible, when the blood in an exposed and unobstructed artery was equally dark as that in the accompanying vein, and when the attempts at respiration were few and imperfect, then before the stop-cock in the trachea was shut, and when the animal was breathing atmospheric air freely. This was so unlooked for, at first sight was so inexplicable, and so much at variance with my preconceived notions on the subject, that I was

* In the experiments first performed the mercury rose in the instrument, but the nitrogen was mixed with a quantity of atmospheric air, as was proved by the blood becoming partially arterialized in an exposed atery.

strongly inclined to believe that there must be some source of fallacy; but after repeating the experiment more than twenty times, and invariably with the same results, I was at last compelled to admit its accuracy. I then began to surmise that this arose from an impediment to the passage of the venous blood through the capillaries of the systemic circulation, similar to that pointed out in the capillaries of the pulmonic circulation, by which the force of the left ventricle was principally concentrated in the arterial system, and on placing a hemadynamometer in the vein of the opposite limb, and comparing its indications with the instrument fixed in the artery, this supposition, as may be seen from the annexed tabular view of the results of one of these experiments, appeared to be verified. This fact may explain how a quantity of blood is retained in the left side of the heart in asphyxia. It was also ascertained that, though the fall of the mercury in the instrument after the animal was nearly asphyxiated took place very gradually at first, it at last fell very rapidly. Suppose, for example, that the mercury in the tube ranged between 4½ and 5 inches in height before the entrance of fresh air into the lungs was prevented, it rose above this when the animal had ceased to struggle; it afterwards fell very gradually to between 3 and 4 inches; and when it had fallen to between 2 and 3 inches, it frequently sunk very rapidly to the original level. When atmospheric air was allowed to enter the lungs after the mercury had sunk low in the instrument, no sooner had the air acted upon the blood in the lungs, than the mercury instantly sprung up several inches, and when the blood had become more perfectly arterialized, it again stood lower, and the range was more limited. The respirations were necessarily much diminished in frequency, also slow and heaving after the stop-cock was opened in an advanced period of the process of asphyxia, and it was remarked, that during each respiratory movement the contractions of the heart were not only performed with increased strength, but likewise with greatly increased frequency. When the animal was breathing freely through the tube in the trachea, was quiescent, and when the blood was fully arterialized, the range of level of the mercury in the tube seldom exceeded half-an inch, sometimes not so much. When the stop-cock was shut no change took place in the range of the mercury during the first half minute: generally before the end of the first minute the animal had begun to struggle, and then the range greatly increased,—rising during each attempt at expiration, and during the struggling of the animal, falling during each attempt at expiration and during quiescence. In some of the experiments the range of the mercury during these different conditions amounted to about nine inches, and in one experiment to ten inches,—making a most material disproportion in

the extent of the pressure upon the inner surface of the arterial system of vessels.

TABLE I.—Showing the changes in inches, of the height and range of the mercurial column in the vertical limb of the haemadynamometer in one of the first class of experiments, when the instrument was fixed in the artery only; the intervals of time at which each change occurred, reckoning in half-minutes from the commencement of the operation; with remarks on the state of the animal at these respective changes. The depth and height of the mercury marked at the end of each half-minute indicated, as near as possible, the extent of the range in the level of the column during that interval of time.

Intervals of Time.	Height of Mercury in the tube attached to the Artery.		Remarks on the state of the Animal.
Minutes.	Depth.	Height.	
		1.0	When the haemadynamometer was adjusted to artery, the mercury stood at this height in the vertical tube of the instrument.
		5.5	At the instant the stop-cock was turned, it was 5.5.
½	4.0	4.5	Stop-cock on trachea shut. Dog quiet.
1	3.0	7.0	Do. The artery becoming a little dark.
1½	3.0	9.0	Do. The artery black. Animal struggling.
2	2.0	12.0	Do. do. Animal struggling violently.
2½	4.0	9.0	Do. do. Animal quiet.
4½	4.0	8.0	Do. do do.
5	4.0	8.0	Stop-cock on trachea opened, and a bladder filled with nitrogen gas applied.
6	3.0	6.0	Do. do.
		11	Bladder of nitrogen removed, and one filled with atmospheric air applied.
7½	5.0	11.0	Bladder removed. Natural respiration allowed.
8½	5.0	6	Do. Animal quiet.

TABLE II.—Showing the same conditions in regard to the second class of experiments, in which haemadynamometers were applied to both the artery and vein at the same time.

Intervals of Time.	Height of Mercury in the tube attached To the Artery.		To the Vein.		Remarks on the state of the Animal.
Minutes.	Depth.	Height	Depth.	Height.	
		0.5		0.0	When the haemadynamometers were adjusted to the vessels, the mercury stood at these heights in the two instruments respectively.
		6.0	5.0	6.0	
½	4.0	5.0		4.0	Respiration natural. Dog quiet.

Interval of Time.	Height of Mercury in the tube attached To the Artery.		To the Vein.		Remarks on the state of the Animal.
Minutes.	Depth	Height.	Depth.	Height.	
2½	3.5	5.0		4.0	Stopcock on trachea shut.
3½	3.0	6.0		3.5	Do.
4	2.0	11.0		12.	Do. animal struggling. The mercury thrown over the top of venous tube, which was 12 inches high.
4½	5.5	10.0		12.	Mercury stood at top of venous tube.
5½	5.5	9.0		8.0	Do.
6½	5.0	11.0		3.5	Do.
7½	5.0	8.		2.5	Do.
8½	2.5			2.1	Do.

In some of the other experiments, the difference between the height of mercury in the two instruments when the blood became venous was not so marked as in this.

In performing these experiments, I derived much valuable assistance from several gentlemen, but more especially from Mr James Spence and Mr K. T. Kemp.

We now proceed to examine the explanations which have been given by physiologists of the cause of the arrestment of the sensorial functions in asphyxia. We have already stated that Bichat maintained that the suspension of the sensorial functions was caused by the circulation of venous blood in the arteries of the brain; while Dr Kay believes that he has proved that it is principally dependent upon a diminished supply of that fluid being sent along the systemic arteries, in consequence of the impediment to the circulation through the lungs, and not because the blood sent to the brain is venous—an opinion somewhat similar to that maintained by John Hunter.* The experiments of Dr Kay, in which he injected, "gradually and gently,"† four drachms of venous blood into one of the four arteries conveying arterial blood to the brain, through a very small syringe, "having a beak with a capillary bore,"‡ though undoubtedly sufficient to prove the highly unsatisfactory nature of the evidence adduced by Bichat in support of his position, that the sensorial functions are arrested by the circulation of venous blood in the arteries of the brain, cannot, however, be adduced as satisfactory evidence against the doctrine itself. Such an experiment may prove that the transmission of a certain quantity of venous blood along *one* carotid artery is not sufficient to produce cerebral derangement; but it cannot enable us to determine what would be the effect of the passage of venous blood along *all the four* arteries of the brain. We have very frequent-

* Hunter's Works by Palmer, Vol. iv. p. 168–170.
† Opus. cit. p. 194. ‡ Opus cit. p. 193.

ly watched an exposed carotid artery in an animal during the process of asphyxia, and have observed that the blood flowing along it gradually becomes darker and darker; and we were satisfied that considerably more venous blood than in the experiments now referred to, is circulated through the brain for a short time before the animal is seized with convulsions and insensibility. It is evident, then, that, if the suspension of the sensorial functions is caused by the presence of dark blood in the arteries of the brain, it must be circulated in greater quantities, and for a longer time than occurred in these experiments of Bichat and Dr Kay. Before we can proceed further in this inquiry, it will be necessary that we examine the variations in the quantity and force with which the blood is sent along the arteries, and returned by the veins during the process of asphyxia. We have already stated that the arterial pressure, as ascertained by the hemadynamometer, is very little altered during the first half minute after the entrance of fresh air into the lungs has been suspended; that about the end of the first, or the beginning of the second minute, when the animal commences to struggle, the pressure is greatly increased; and that, generally, for about two minntes after the animal had become insensible, and had consequently ceased to struggle, the pressure was even greater than before the commencement of the experiment. It was also repeatedly ascertained, that the venous pressure, as indicated by the hemadynamometer introduced into the jugular and femoral veins, was equally great for a short time after the animal had become insensible as before the respiration had been suspended. When an artery is cut across, immediately after insensibility has supervened, the blood springs from it in a full stream, and with a force equal to what would occur if arterial blood was circulating in the vessels. The insensibility in asphyxia cannot therefore depend upon any diminution in the force with which the blood is sent along the arteries of the brain, nor upon any diminution in the vascular pressure upon that organ. As, however, the frequency of the pulsations in the arteries becomes remarkably diminished before the circulation has been fairly suspended, we are naturally led to inquire if any change in the quantity of blood sent along the arteries of the brain could account for the suspension of its functions. With this view, we performed several experiments upon dogs. A tube furnished with a stop-cock was introduced into the trachea, and firmly secured there, the femoral artery was then laid bare, that the changes in the blood might be observed and the number of pulsations more carefully reckoned. We shall give the details of four of these experiments. After the femoral artery had been laid bare, the pulse ranged from 105 to 120 in a minute, and the respirations were very short and rapid. At the end of the first half-minute after the

stop-cock was turned, the pulse was 92. At $1\frac{1}{2}$ minute the pulse was about 120, the animal had begun to struggle, and the blood in the artery was decidedly dark. At the second minute the blood in the artery was nearly as dark as in the accompanying vein, but, from the struggles of the animal, it was impossible to reckon the pulse. At the end of $2\frac{1}{2}$ minutes the animal had ceased to struggle, was evidently insensible, and the pulse was 42. At the beginning of the fourth minute, the pulse was still 42. The stop-cock was now opened, and the animal allowed to breathe. When the blood was becoming red in the artery, the pulse was 78. A short time after this, when the animal was rapidly recovering its consciousness, the pulse was 60, and the respirations about 132. In another experiment the pulse was 80 at the time when the stop-cock was closed. At the end of the first minute the pulse was 114, and the blood was decidedly darker, and the animal was struggling. At the $1\frac{1}{2}$ minute the animal was struggling, and the blood was nearly as black as in the accompanying vein. At the end of $2\frac{1}{2}$ minutes the pulse was 60, irregular in frequency—two beats following each other rapidly; the animal had ceased to struggle, and the blood was as dark as in the vein. At the end of the third minute the pulse was still 60, and irregular. In a third experiment the pulse was 100 before the stop-cock was turned. At the end of one minute the blood was getting dark, the animal had begun to struggle, and the pulse was 120. During the course of the second minute it struggled violently, and the pulse could not be reckoned. At the end of $2\frac{1}{2}$ minutes the animal had ceased to struggle, the respirations were few and heaving, and the pulse was 78. At the end of the third minute the pulse was 60. In a fourth experiment the pulse ranged from 88 to 96 before the stop-cock was turned. After half-a minute the pulse was 71, and the blood was somewhat darker. After $2\frac{1}{2}$ minutes the animal had ceased to struggle, the blood was as dark in the artery as in the vein, and the pulse was 70. At the end of the third minute the efforts at breathing had nearly ceased, and the pulse was 66. In such experiments as these, it is impossible to ascertain the exact frequency of the pulse at the precise moment when the sensorial functions are suspended, in consequence of the struggles and convulsive movements with which this is preceded. Taking, however, all the circumstances of the experiments into account, and combining with them the facts ascertained in those previously detailed, to prove that the arterial and venous pressure is not diminished at the time that the animal has become insensible, we have little difficulty in arriving at the conclusion, that, though the pulse has become less frequent about the time that the insensibility has supervened, yet that this has not taken place to such an extent as to justify the opinion, that the arrest-

ment of the sensorial functions depends upon any diminished transmission of blood through the vessels of the brain. If a diminution in the frequency of the pulse to the extent we have indicated could produce insensibility, this would frequently present itself during the course of disease, and under other circumstances where nothing approaching to it is observed. It must also be remembered that the pulse, as ascertained before the experiment had been commenced, must have been more frequent than usual, from the terror of the animal. In these experiments I regarded the animal as in a state of insensibility when the struggles and convulsive movements had ceased. The function of respiration continued for a short time after the suspension of the sensorial functions, but rapidly became enfeebled. The circulation of the dark blood in the vessels of the encephalon, therefore, arrests the functions of the cerebral hemispheres before those of the *medulla oblongata*.

Dr Kay has performed several experiments,* from which he has drawn conclusions very different from those which we have just stated. He found, that when the abdominal aorta was cut across in a rabbit of the ordinary size, " nearly seven drachms and three-quarters of blood would escape from the divided aorta when respiration was unobstructed." He then proceeded to cut this vessel across at different periods after the admission of fresh air into the lungs was precluded, and found that, when cut across half a minute after this, the blood collected almost equalled what would have escaped if the free access of air into the lungs had been permitted. In another animal it was cut across after a minute and a half, and five drachms of blood escaped ; when postponed to two minutes and a-half, four drachms were collected ; and when delayed to the termination of the third minute, only two drachms were collected. In judging of the value to be attached to these experiments of Dr Kay, two circumstances are to be taken into account—the time an animal requires to bleed to death ; and the precise time at which the sensorial functions are arrested. As there can be no doubt that an impediment to the circulation through the lungs does occur in the course of the process of asphyxia, it is, therefore, a matter of considerable importance to ascertain not only the precise time at which the sensorial functions are arrested, but also the average period of time which the blood would continue to flow from a cut artery when the respiration is unobstructed, before we can venture to determine whether there is any relation between the suspension of the sensorial functions and the arrested circulation in the lungs. With the view of satisfying myself on these points, the abdominal aorta in a rabbit breathing naturally was cut across a little above its bifurcation. The blood continued to flow freely for

* Opus cit. p. 185—88.

about one minute; it flowed feebly for another minute; and very feebly for about forty seconds more. In this experiment two minutes and forty seconds elapsed before the bleeding from the artery had ceased. Though in some subsequent experiments the hemorrhage had ceased in a somewhat shorter time, yet we believe that in the rabbit it seldom stops before two minutes have elapsed. With regard to the other point we have mentioned, viz. the exact period at which the sensorial functions are arrested, this has been most unaccountably overlooked by Dr Kay. He seems not to have been aware that a dog generally becomes insensible in from two to two and a-half minutes, and a rabbit in one minute and a-half after the complete occlusion of air from the lungs, so that experiments such as those he has related, made to ascertain the quantity of blood which flows from a cut artery at periods posterior to the occurrence of the suspension of the sensorial functions, cannot be adduced in explanation of effects which have previously happened. In performing the experiments as I have already mentioned, I took the cessation of the struggles and the convulsive movements of the animal as a test of insensibility. When a ligature is tied tightly around the trachea of a rabbit, the animal moves about nimbly at first, but, before one minute and a-half have elapsed, it has fallen down in a state of insensibility, and the attempts at respiration are few and heaving. As the manifestation of the functions of the *medulla oblongata*, upon which respiration depends, are not necessarily linked with that of the functions of the cerebral hemispheres or the sensorial functions, it must be evident that, in attempting to discover the cause of the cessation of the mechanical movements of the chest, the frequency of the respirations ought to be attended to, and not the suspension of the sensorial functions. This circumstance has not been overlooked by us in performing these experiments; and we are satisfied that the function of respiration is much enfeebled at a period of the process of asphyxia, when this cannot be explained by any diminution in the quantity of blood sent to the *medulla oblongata.* No doubt respiratory movements may be observed after the pulsations have been very considerably diminished in frequency, but these have become few in number, and performed at long intervals before this condition of the circulation has been induced; but it is quite possible that the ultimate cessation of the functions of the *medulla oblongata* may be hastened by the diminished quantity of blood sent along the arteries supplying it. If we proceed, therefore, to analyse the experiments of Dr Kay, bearing in mind the length of time the blood continues to flow from the divided abdominal aorta of a rabbit, and the precise time at which the sensorial functions are arrested in the process of asphyxia, we must arrive at very different conclusions from those which he has deduced from them.

In further confirmation of the views we are advocating, we may

appeal to the experience of every practical physician ; for he cannot have failed to observe the gradual torpor that frequently creeps over the sensorial functions in severe cases of bronchitis, when an ill-arterialized blood is circulating in the vessels of the brain, and the pulse is still pretty strong at the wrist.

We feel very strongly convinced that Dr Kay has fallen into another error in stating, that, three minutes after the entrance of air into the lungs had been prevented, the blood in the arteries had assumed the venous hue " still imperfectly ;" for, in numerous experiments, various gentlemen who were present all agreed that the colour of the blood in the arteries was as dark as that contained in the accompanying veins at a period anterior to this. The statement of Bichat, that the blood in the arteries exactly resembles venous blood in a minute and a-half or two minutes, is, I am satisfied, much nearer the truth.

From the various facts we have mentioned, we have arrived at the conclusion, that the suspension of the functions of the encephalon are chiefly, if not entirely, dependent upon the circulation of venous blood in the arteries. We do not, however, maintain that venous blood exerts any noxious influence upon the functions of the nervous texture ; but believe that the effects are solely to be attributed to the want of the proper excitation of the organ ; for, when the circulation of arterial blood is renewed, its functions rapidly remanifest themselves, provided that this be done within a given time.

We believe, then, that, in asphyxia, the order of succession in which the vital processes are arrested is as follows ;—The venous blood is at first transmitted freely through the lungs, and reaches the left side of the heart, by which it is driven through all the textures of the body. As the blood becomes more venous, its circulation through the vessels of the brain deranges the sensorial functions, and rapidly suspends them, so that the individual becomes unconscious of all external impressions. The functions of the *medulla oblongata* are enfeebled about the same period that the sensorial functions are arrested, but are not fairly suspended for some time longer. Immediately after the sensorial functions are suspended, and the blood has become still more venous, it is transmitted with difficulty through the capillaries of the lungs, and consequently begins to collect in the right side of the heart. A smaller quantity of blood must now necessarily reach the left side of the heart ; and this diminution of the quantity of blood sent along the arteries, conjoined with its venous character, and the ultimate arrestment of the circulation, being circumstances incompatible with the manifestation of vitality in the other tissues of the body, general death is sooner or later induced.

The persistence of the muscular contractility after the arrestment of the circulation varies, as we have had frequent opportunities of

witnessing, according to the age and strength of the individual, and also in a very marked manner from constitutional causes, which are unknown; and in this way we are able to explain how the heart's action may be renewed a considerable time in some cases after apparent death, while in others all the attempts to restore animation, though commenced shortly after the suspension of the sensorial functions, have failed. It must be obvious that the first and principal object in the treatment of asphyxia is to restore the circulation through the lungs. If once we succeed in this, and thus renew the heart's action, the arterial blood is again transmitted to the encephalon and the other tissues of the body; the functions of the *medulla oblongata* remanifest themselves; the sensorial functions are gradually restored; and the animal heat returns. The derangement of the functions of the *medulla oblongata* and the sensorial functions are not necessarily coequal in extent, and never in importance, in asphyxia, and this is well observed in some of those cases of death from disease or narcotic poisons, where the process of asphyxia occurs more slowly and gradually. In these it is not unusual to find the sensorial functions nearly or entirely suspended, at a time when the respiration is pretty effectively carried on; and it is evident from various facts, that the arrestment of the muscular respiratory movements is not dependent upon the suspension of the sensorial functions, but upon those of the *medulla oblongata.*

We shall now proceed to make some remarks upon the increased force with which the blood is sent along the arteries during muscular contraction. It has been proved, as we have already mentioned, that the blood is sent with greater velocity and increased force along the arteries during the contraction of the muscles of the limbs and trunk, as in exercise, and this takes place in a more marked manner during violent attempts at expiration. On the other hand, during violent attempts at inspiration, the pulse becomes less frequent, feeble and soft. In some of the experiments we performed, as we have already mentioned, the mercury rose as high as the eleventh, and in one to the twelfth inch of the scale attached to the tube, during violent attempts at expiration, and the struggles of the animal; while it fell as low as the second inch during violent attempts at inspiration. During these different conditions, the pressure upon the external surface of the heart and its position in the chest must be somewhat altered, a certain amount of pressure being applied to its outer surface during expiration, and removed during inspiration; and it recedes deeper into the chest during inspiration, and again comes forward during expiration;* but we may safely set those aside as exerting any

* In mentioning this fact in the article Heart, in the Cyclopedia of Anatomy and Physiology, the word inspiration has been inadvertently printed for expiration, and *vice versa.*

appreciable influence in the production of the phenomena in question. Müller believes that the increased contractions of the heart accompanying muscular movements of the trunk and limbs may be caused by a sympathetic or reflex action,—an excitant effect being produced in the filaments of the nerves distributed in the contracting muscles, which, being conveyed inwards to the spinal cord, is reflected upon the heart. As, however, he adduces no direct evidence in favour of this opinion, we do not feel inclined to abandon the old explanation, that this is merely dependent upon the mechanical acceleration of the blood, by the pressure exerted upon the blood-vessels by the surrounding muscles during their contraction, and the more especially, as we have witnessed several facts, which at least prove that a great part of the phenomena in question may arise from this cause. We have frequently remarked, that when an animal was breathing very rapidly, even above 100 in a minute, through a tube in the trachea, that the mercury did not rise higher in the instrument than before, and that the range was limited, provided the expirations were always short, and, consequently, not attended with much compression of the blood-vessels in the thorax and abdomen. On the other hand, a marked rise of the mercury took place whenever a forced expiration was made, however slowly this was performed. It was also repeatedly observed, that when one instrument was fixed in the femoral artery, and another in the femoral vein of the opposite limb, the mercury stood considerably higher in the instrument fixed in the vein than in that fixed in the artery, when the animal began to struggle violently. In few of the experiments did the mercury rise much above eleven inches in the instrument in the artery, while it frequently ran over the top of a tube twelve inches high, with considerable force, in the instrument fixed in the vein, —showing us in some of these experiments a prodigious increase in the pressure upon the inner surface of the venous system, equal to between three and four pounds on every square inch of surface. This greater elevation of the mercury in the instrument fixed in the vein, can only be explained by the effects of the mechanical pressure of the surrounding muscles becoming increased, as the extent of the vascular tubes over which it is exerted becomes elongated, and may afford some indications of the greatly increased impulse communicated to the blood by the powerful pressure exerted by the contraction of the muscles of the chest and abdomen upon their contained blood-vessels, when aided by the contractions of the muscles of the limbs, and favoured by the presence and particular disposition of the valves of those blood-vessels. It is difficult to determine, then, how much this increased flow of blood along the vessels during violent expirations, and during the

contraction of the muscles of the limbs, depends upon more forcible contractions of the heart, or upon the mechanical effects of temporary pressure upon the blood-vessels. The increased rapidity and strength of the contractions of the heart during violent expirations must be partly attributed to the compression of the blood-vessels of the lungs, and the transmission of an increased quantity of blood to the left side of the heart, while the diminution in the strength and frequency of the pulse during inspiration must, in a great measure at least, depend upon the sudden removal of that pressure, so that a great part of the blood propelled during a few of the contractions of the right side of the heart, which immediately succeed the sudden dilatation of the thorax, goes to fill up the blood-vessels of the lungs to that state of plenitude in which they were before the preceding expiration, and a small quantity only reaches the left side of the heart.

We do not think it necessary to make any remarks upon the question, whether or not the blood stagnates in the lungs, in consequence of the cessation of the chemical changes between the blood and the atmospheric air, or upon any supposed effect which the venous blood may have upon the contractility of the capillary vessels of the lungs, as this has already been most ably and most satisfactorily done by Dr Alison. He has shown that this phenomenon is to be referred to an interesting general law in physiology, which has hitherto not received the attention which its importance demands, by which the movements of nutritious juices is influenced by the chemical changes, or, as he terms them, the vital attractions connected with the chemical changes, which are constantly going on in the capillary vessels between those juices and the surrounding tissues, by which nutrition and secretion are effected. That such a moving power exists, regulating the quantity of blood which flows through each individual organ, independent of any impulse from the living solids, cannot be doubted.* Before arterial blood can be transmitted freely through any tissue or organ, it is not only necessary that the contractions of the heart be performed with a certain amount of force, but that the actions of nutrition and secretion be also in operation ; so in the same manner, before the blood can be transmitted through the lungs, it is not only necessary that the right side of the heart retains its contractility, but that the chemical changes between the blood and the atmospheric air should proceed. This doctrine is still further illustrated by the fact which we have ascertained, that, when the blood in the systemic circulation becomes decidedly venous, and unfit for carrying on the process of nutrition, it passes less freely through the capillary arteries into the veins.

* Vide Outlines of Physiology, 3d edition, p. 22-25, 61-64, and 224.

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